January 8, 1999

This document was submitted to EPA by a registrant in connection with EPA's evaluation of this chemical, and it is presented here exactly as submitted.

December 8, 1998

Mr. Angel Chiri
Special Review and Reregistration Division
U.S. Environmental Protection Agency
401 M Street, SW
Washington, DC 20460-001

SUBJECT: Response to the Draft EFED and HED Reregistration Eligibility Decision (RED) Science Chapters for Methamidophos, List A Case 0043

Dear Mr. Chiri:

Following is a response to the draft Environmental Fate and Effects Division (EFED) and Health Effects Division (HED) chapters for methamidophos which BAYER received from the EPA on November 10, 1998. Comments regarding the chapters and our plan for continued support for methamidophos are as follows:

Both chapters contain preliminary, screening-level calculations based on all "current" methamidophos labels. The review is complicated somewhat by the fact that methamidophos registrations and tolerances are influenced by the use of acephate as well, since acephate degrades into methamidophos as it breaks down in the environment. Acephate is not registered by BAYER Corporation, but relevant information is coordinated with Valent, the primary registrant of acephate.

Overall, the document describes the state of the reregistration efforts for methamidophos and with few exceptions, indicates that the current database is adequate to support continued registration of the chemical. The document goes on to indicate that there are no carcinogenicity concerns for methamidophos (classified as GROUP E), nor was there any evidence for increased susceptibility of methamidophos to infants or children. Finally, the document indicates that the additional 10X safety factor could be removed or lowered to 3X based on the toxicity data available for methamidophos. BAYER has found several areas which could be classified as errors of omission relative to the assessment of risk and are outlined below:

Errors to be Considered in the EFED Chapter

Drinking water was not addressed in the RED because it was indicated that the acute risk cup was "filled" with dietary contributions. However, an aquatic exposure assessment was performed to support the ecotoxicology risk assessment. These first comments are directed toward that assessment:

1. PRZM/EXAMS exposure modeling did not include the default assumption where an

aerobic aquatic metabolism half-life is set to 2x the aerobic soil when an acceptable study does not exist. In this case an aerobic soil half-life of 1.75 days would be used, making the estimated aerobic aquatic half-life at 3.5 days. References for the use of 2x the aerobic soil rate are:

"Input Selection for Computer Modeling of Aquatic Pesticide Exposure Using the PRZM2 and EXAMS II Programs" Version 1.1, June 1995. USEPA/OPP

"Guidance for estimating metabolic degradation input parameters for GENEEC, PRZM and EXAMS when estimating exposure in surface water. Draft document by R. David Jones (EPA), April 28, 1998.

Impact on the assessment: The aquatic values used in the ecotoxicology assessment are overestimated due to a lack of chemical reactivity in the farm pond. The exposure assessment also cannot be used in a reliable drinking water exposure assessment.

It should be noted however, that the EPA indicated that methamidophos should not be a groundwater concern due to the short-lived nature of methamidophos and its degradates in/on aerobic soil.

2. The draft RED for methamidophos contains several erroneous and misleading statements in its summary of the avian field studies of Blus et al. 1989 (p 31-32 and p 44-45), Perritt et al. 1990 (p 31 and p 47-48). The specific errors and the correct information for each case are presented below:

   a. Field Study of Blus et al. (1989) – in this study, the draft RED refers to a large field kill incident of sage grouse documented by these authors in Idaho in 1986. A number of erroneous and misleading statements concerning this study are included on p 31-32 and p 44. The draft RED states on p 31-32 the following:

      "One hundred sick or dead grouse were observed around 2 potato fields in 1986 after spraying with methamidophos in potato fields and dimethoate in alfalfa fields. Most of the sage grouse died in or near the fields and some of the grouse were tracked in the sagebrush up to 1 Km from the fields."

      On p 44, it is further stated under the heading "<u>Field Study and Incidents Show Extensive Population Risk</u>":

      "Field studies over time (Blus et al., 1989) showed that significant sage grouse populations near potato fields can be killed by the use of methamidophos. Data collected in 1983 show brain ChE depressions of 40-65% in sage grouse collected near potato fields shortly after spraying with methamidophos. One hundred

intoxicated or dead grouse were observed around 2 potato fields in a follow-up study in 1986 with radio-collared sage grouse after the potato fields were sprayed with methamidophos."

The above statements imply that the study of Blus et al. (1989) documented methamidophos as a major cause of sage grouse mortality, with up to 100 birds intoxicated in a single incident and population level impacts demonstrated. In actuality, nearly all pesticide-related mortality of sage grouse documented in the 1985-1986 field study of Blus et al. (1989) was attributed to the use of dimethoate on alfalfa, and NOT methamidophos on potatoes. In this study, 82 healthy grouse were radio-collared prior to insecticide treatments. Fourteen of these birds (17%) became intoxicated from insecticide exposure, and 9 of them (11%) died. Seven of these deaths (9%) were attributed to dimethoate intoxication. For 2 (2%) of the radio-tagged grouse, methamidophos was implicated as a contributing cause of death. Even in these two cases, forensic analysis (brain ChE assay and residues in GI tract contents) did not conclusively confirm methamidophos as a cause of death. Brain ChE inhibition of these two birds were 39 and 43%, respectively. This is below the 50+% inhibition criteria for concluding death was caused by exposure to an organophosphate. The GI tract of one of the two birds contained 18 ppm methamidophos, which is only about 40% of the lowest avian LC50 value. Additionally, the carcasses had been partially eaten and buried, a finding that suggests death was the result of predation by a coyote. It is possible that Methamidophos intoxication may have increased the vulnerability of these two grouse to predation, however, even this conclusion is speculative.

The major bird kill incident in this study occurred in 1986 at a 240 ha alfalfa field sprayed with demethoate. A flock of 200 sage grouse occupied this field when it was sprayed on 1 August. Over the next 12 days, 63 dead sage grouse were found here including 5 of the radio-tagged population. Forensic evidence confirmed dimethoate as the cause of death of many of these grouse. Contrary to the statement in the draft RED, methamidophos was NOT implicated in this major incident. The draft RED is therefore inaccurate in its statement that this study "showed that *significant* sage grouse populations near potato fields can be killed by the use of methamidophos."

b. Field Study of Perrit et al. (1990). On p 31, the draft RED states:

"There were 24 deaths after treatment at this plot with 2 mammals, 1 reptile and 2 amphibians containing methamidophos residues.....The study was considered supplemental because the cause of 17 avian deaths could not be determined; however, methamidophos could not be precluded as the cause of death."

And on p 47-48, the draft RED states:

"A cotton field residue study (MRID 41548803) reported 17 deaths after treatment with 2 mammals, 1 reptile, and 2 amphibians having methamidophos residues. Of the carcasses placed at the site, 30% were found. Given carcass removal/detectability rate (17/30% = 57), a total number of 57 casualties could not be precluded as being treatment-related."

The above statements erroneous imply that (1) a single field plot was studied, and (2) 17 avian deaths were documented for which methamidophos could not be precluded as a cause of death. In actuality, 8 cotton fields in east central Alabama were studied. Four avian deaths were documented on these fields after application. All four of these were scavenged carcasses consisting mostly of feathers ("feather spot"). Methamidophos could neither be attributed or ruled out as cause of death of these individuals. However, the fact that the number of avian mortalities recorded at these sites prior to the first methamidophos applications (3) was about the same as afterwards (4) suggests that methamidophos had a minimal effect on avian survival in this study.

Errors to be Considered in the HED Chapter

No specific errors were noted in this chapter other than errors of interpretation. BAYER does not agree with the MOE's used in the assessment provided and will provide additional data in the public comment period which will address the appropriate safety factors to be used. BAYER believes that the calculated risks of methamidophos use are acceptable but continues to generate new information which will be used to resolve any concerns there may be regarding the safety of methamidophos use. To further this point, these issues are on the top of BAYER's agenda, and, in the last year, BAYER has proposed several measures to further reduce exposure to methamidophos, and expand confidence in the supporting database. In addition, we continue to develop new data to address requirements of the Food Quality Protection Act (FQPA). Some of those measures and data include:

1. Participating in the Best Management Practices Group educational program entitled "SAFETY: Apply it First", which was aimed at reminding growers and distributors that some extra attention should be used when using organophosphate pesticides.
2. Distributing by the 1999 use season, all of BAYER's methamidophos products in closed-delivery systems, including its liquid products sold in 2.5 gallon containers. This proposal necessitated working with an equipment manufacturer to develop such a system which has been favorably reviewed and received by the Agency.
3. Including additional label language to lessen the opportunity for exposure from spray drift.
4. Voluntarily removing several crops from methamidophos labels which no longer have support from grower groups or other registrants.
5. Initiating additional residue and use studies to evaluate residue levels on select crops given current crop and pest management programs.

6. Developing additional exposure and toxicology data to refine occupational and dietary assessments, this will include a Monte Carlo assessment.

As this new information becomes available, BAYER will continue to work with the Agency to refine the risk assessment further for methamidophos. Additionally, BAYER will be looking to the Agency for assistance in developing methodology for considering the contribution from acephate as well as methamidophos in calculating the risk from the residues of these agricultural tools.

In conclusion, BAYER stands behind the safety of methamidophos under current use conditions. We will continue to work with the Agency and User Community to address issues and concerns raised by the Agency or FQPA. If you have any questions, please contact either me or Dr. James Kunstman at (816) 242-2838.

BAYER CORPORATION
AGRICULTURE DIVISION

John S. Thornton
Director, Product Registrations &
Regulatory Affairs

cc: Marilyn Mautz